REMARKS

Favorable reconsideration is respectfully requested in view of the foregoing amendments and the following remarks.

I. CLAIM STATUS AND AMENDMENTS

Claims 1-16 were pending in this application when last examined and stand rejected.

Claims 1-8 are cancelled without prejudice or disclaimer thereto. Applicants reserve the right to file a Continuation or Divisional Application on any cancelled subject matter.

No new matter has been added.

II. WRITTEN DESCRIPTION REJECTION

On pages 2-3 of the Office Action, claim 1 was rejected under 35 U.S.C. § 112, first paragraph, as failing to comply with the written description requirement. Without acquiescence to the correctness of this rejection, claim 1 is cancelled and therefore this rejection is moot.

III. ANTICIPATION/OBVIOUSNESS REJECTIONS

On pages 3-4 of the Office Action, claims 1-3 and 9-11 were rejected under 35 U.S.C. § 102(b) as anticipated by Exton et al.

Further, on pages 4-6, claims 1-16 were rejected under 35 U.S.C. § 103(a) as obvious over Exton et al. in view of Williams et al. and further in view of Sharif et al.

Applicants respectfully traverse these rejections as applied to the remaining claims. Applicants further note that claims 1-8 have been cancelled without acquiescence to the correctness of the Examiner's position and therefore in regard to these claims, the above-noted rejections are moot.

With regard to pending claims 9-16, Applicants provide the following remarks.

Exton et al. describes that anorexia is induced by Prostaglandin E_2 (PGE_2) (see page 471, left column, lines 17-18). Also, it is mentioned that lipopolysaccharide (LPS) induced anorexia is attenuated by indomethacin (see page 471, left column, lines 18-19). In this regard, it was known to those skilled in the art that LPS induces arachidonate cascade, which leads to the production of PGs, while indomethacin inhibits cycloxygenase, which leads to blocking of

arachidonate cascade to attenuate PGs production. Accordingly, those skilled in the art understand that Exton et al. discloses that anorexia can be induced by the production of PGs and that anorexia can be attenuated by the reduction of the production of PGs.

Additionally, Exton et al. concludes that LPS-induced anorexia is PG dependent due to retardation of gastric emptying mediated by PGs (see page 475, right column, lines 41-46).

Thus, Exton et al. suggest increased production of PGs to lead to inhibition of food intake and reduction of PGs production to lead to recovery or stimulation of food intake.

In contrast with Exton et al., the claimed inventions are directed to a method for stimulating food intake by the administration of PGD_2 (or PGD_2 agonist) and a method for inhibiting food intake by the administration of PGD_2 antagonist. That is, the claimed inventions are based on the discovery that food intake can be stimulated by enhancement of the action of PGD_2 and inhibited by attenuation of such action. This is in direct contradiction to the teachings of Exton et al.

Consequently, claims 9-11 are not anticipated over Exton et al., which discloses the role of prostaglandin contrary to that of the claimed inventions.

Further, the claimed inventions are not obvious over Exton et al. in combination with Williams et al. and/or Sharif et al. as such references fail to remedy the noted deficiency of Exton et al.

Thus, Applicants respectfully suggest that these rejections are untenable and should be withdrawn.

CONCLUSION

In view of the foregoing amendments and remarks, it is respectfully submitted that the present application is in condition for allowance and early notice to that effect is hereby requested.

If the Examiner has any comments or proposals for expediting prosecution, please contact the undersigned attorney at the telephone number below.

Respectfully submitted,

Masaaki YOSHIKAWA et al.

/William R. Schmidt, II/

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